

the decade they investigated, 232 (7.7%) were schizophrenic and eight (0.3%) were suffering from affective disorders. (The corresponding proportions for women were 6.4% and 3.6% respectively.) These figures are close to the ones found here ($\chi^2=0.26$ for people with schizophrenia and 0.99 for those with affective disorders), which suggests that in Europe 5-10% of homicidal violence committed by men is perhaps accounted for by schizophrenia and $\frac{1}{2}$ -1% by depression.

At the beginning of this report we emphasised that any sample of violent and disturbed men might be representative of nothing more than that sample. Certainly these risk findings cannot be widely generalised, but in Europe the risk of schizophrenic violence should not now be underestimated. This may be related to current policies of discharging patients with schizophrenia from hospital or restricting their admission without, in many cases, the provision of adequate alternative care. Continuing to make insufficient provision may have increasingly serious consequences.

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Acute renal failure due to rhabdomyolysis associated with use of a straitjacket in lysergide intoxication

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Abstract

Acute renal failure is a known sequel to rhabdomyolysis, both traumatic and non-traumatic. Two patients who had been placed in straitjackets after taking lysergide (LSD) developed acute renal failure and rhabdomyolysis. One subsequently died.

The rhabdomyolysis probably resulted from a combination of severe restraint and the violent movements induced by the drug. The use of straitjackets cannot be considered to be completely safe in such cases.

Introduction

Rhabdomyolysis due to trauma, so called crush injury, is a well recognised cause of acute renal failure.¹ Non-traumatic rhabdomyolysis may cause acute renal failure after fits, prolonged coma, and viral myositis² and is also associated with overdoses of heroin, alcohol, and barbiturates.³ We describe here two patients who had become violent after taking lysergide

(LSD) and had been placed in straitjackets before admission to hospital. Both developed acute renal failure and rhabdomyolysis, as shown by a considerable rise in serum creatinine phosphokinase and uric acid concentrations.

Case 1

A 19 year old white man became violent after taking lysergide, necessitating the use of a straitjacket. He was taken to the local hospital, where he was easily sedated with chlorpromazine. He had a right orbital haematoma and superficial cuts to the wrists and ankles with many superficial abrasions over his back. Over the next four days he became oliguric with rapidly rising plasma urea and creatinine concentrations. He was transferred to Charing Cross Hospital, where peritoneal dialysis was started. Initial investigations showed extremely high values of plasma creatine phosphokinase (45 000 IU/l, normal <90 IU/l), alanine transaminase (1690 IU/l, normal <35 IU/l), and urate (1155 μ mol/l (19 mg/100 ml), normal 100-400 μ mol/l (1.7-6.723 mg/100 ml)). Rhabdomyolysis was therefore diagnosed, and the findings on renal biopsy suggested acute tubular necrosis. Over the next few weeks his renal function spontaneously improved and he was eventually discharged with normal renal function. Two years later his renal function remains normal.

Case 2

A previously fit 25 year old white man had become violent after trying to jump off a roof having taken lysergide. He was restrained in a straitjacket and brought to Charing Cross Hospital at

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7.30 pm. On arrival he had hyperpyrexia (41.6°C), hypotension (80/40 mm Hg), and generalised muscle twitching. Sedation was extremely difficult despite large doses of intravenous haloperidol and chlorpromazine, and he was eventually given sodium thiopentone. He remained feverish for some hours. Overnight he passed only small amounts of dark urine and by the next morning his plasma urea and creatinine concentrations had risen to 17.5 mmol/l (105 mg/100 ml) and $500\text{ }\mu\text{mol/l}$ (5.7 mg/100 ml) respectively. Plasma creatine phosphokinase ($13\,760\text{ IU/l}$), alanine transaminase (1100 IU/l), aspartate transaminase (1715 IU/l), and urate ($1230\text{ }\mu\text{mol/l}$ (20.7 mg/100 ml)) were all raised. He remained hypotensive, and after three cardiac arrests he died at 11.45 am.

The disproportionately high plasma creatinine concentration for such a short period of renal failure and the enzyme results suggested extensive rhabdomyolysis. At necropsy he was noted to have multiple superficial cuts and abrasions and areas of bruising; the tissue beneath these areas showed extensive haemorrhage and bruising. Examination of the kidneys showed extensive casts in the tubules compatible with myoglobin.

Subsequent toxicological analysis revealed an extremely high plasma lysergide concentration before death of 14.4 ng/ml .

Discussion

Both these patients appeared to develop rhabdomyolysis after being in a straitjacket because they became violent after taking lysergide. There is no evidence that lysergide is directly

nephrotoxic, although, as with fits, the violent movements caused by the lysergide itself might have resulted in rhabdomyolysis. It is more likely, however, that the rhabdomyolysis was caused by muscle injury related to the restraint imposed by the straitjacket. Straitjackets are rigid, and extensive superficial and muscle injuries might be caused when they are used to control violent patients. There are no other reports of straitjackets causing rhabdomyolysis despite their use for decades. Nevertheless, patients who have taken lysergide may become very violent, and possibly the combination of extreme violence and severe restraint precipitated the onset of rhabdomyolysis in these patients. Thus, although it can be difficult to control such patients, even straitjackets cannot be used without risk.

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Release of β endorphin and met-enkephalin during exercise in normal women: response to training

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Abstract

Plasma β endorphin and met-enkephalin concentrations were measured in response to treadmill exercises in 15 normal women before, during, and after an intensive programme of exercise training. Significant release of β endorphin occurred in all three test runs, and the pattern and amount of release were not altered by training. Before training dramatic release of met-enkephalin was observed in seven subjects and smaller rises observed in a further four, and this response was almost abolished by training. This represents the first observed "physiological" stimulus to met-enkephalin release.

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Endogenous opioid peptides play a part in adaptive changes to exercise training and probably contribute to the menstrual disturbances of women athletes.

Introduction

Exercise training is becoming increasingly popular in both Great Britain and the United States for the prevention and treatment of disease, over 20 000 people having competed in a recent London marathon alone. For women, however, regular strenuous physical exercise may result in several menstrual disturbances, including delay of the menarche,¹ secondary amenorrhoea,² and inadequate luteal phase.³ These women have been reported to have low circulating gonadotrophin concentrations with abnormal pulsatility and exaggerated responses of gonadotrophin during a luteinising hormone releasing hormone test,⁴ suggesting that the amenorrhoea is mediated at the hypothalamic level. Endogenous opioid peptides inhibit pulsatile gonadotrophin release⁵ and have been implicated in these exercise induced changes, as infusion of the opiate antagonist naloxone causes a striking increase in amplitude of luteinising hormone and follicle stimulating hormone pulsations in amenorrhoeic runners.⁴

The opioid peptide β endorphin and its immediate precursor lipotrophin are both derived from pro-opiomelanocortin⁶—the precursor of adrenocorticotrophic hormone—and are released from the anterior pituitary in response to "stress," including exercise.⁷⁻¹² A previous study suggested that the response of β endorphin and β lipotrophin is facilitated by exercise training,⁹ and this has been suggested as the biochemical basis of the